effective in clearing Aβ plaques in *in vivo* and *ex vivo* experiments with PDAPP mice.

In paragraph 24 of the Official action, the examiner contends that the specification does not provide sufficient guidance that would enable the skilled artisan to conceive of and make any antibody that would prevent or reduce aggregation or disaggregate aggregates in a subject.

In view of the amendments to the claims (new claims 150-167), which recite that the epitope is within amino acids 1-28 of Aβ, or is obtainable using 1-28 of Aβ as the immunogen, applicant respectfully submits that the examiner's rejection has been rendered moot.

In paragraph 25 of the Official action, the examiner contends that undue trial and error experimentation would be required to make antibodies that are capable of prevention or reduction of Aβ aggregates or disaggregate the same in patients.

Contrary to the examiner's contention, as discussed above, the present specification shows that AMY-33 (raised against Aβ amino acids 1-28) inhibits aggregation of Aβ, Hanan et al (1996) shows that 6C6 and 10D5 (both raised against amino acids 1-28) inhibit aggregation of Aβ, and Solomon (PNAS 1997) shows that 6C6 (raised against amino acids 1-28) causes disaggregation of Aβ aggregates. On the other hand, the evidence shows that 6F/3D (raised against Aβ amino acids 8-17) does not inhibit Aβ aggregation. It would clearly not require undue experimentation for one skilled in the art to produce

antibodies with the claimed specificity (which the post-filing evidence (Bard et al (2000); Bard et al (2003); Bacskai et al (2001); and DeMattos et al (2001)) clearly demonstrates are effective at inhibiting aggregation of Aβ and disaggregating Aβ aggregates). That is, one could merely use, e.g., Aβ 1-28 as an immunogen, and assay for inhibition of aggregation or disaggregation, as described in the present application.

In paragraph 27 of the Official action, the examiner cites Walker et al for teaching that the anti-Aβ antibody 10D5 did not disaggregate, prevent or inhibit aggregation.

Applicant respectfully submits that the examiner has mischaracterized Walker et al. Walker et al merely relates to *in vivo* imaging of Aβ deposits in the brain. Walker et al did not look for, much less carry out any experiments to measure disaggregation or prevention/inhibition of aggregation. In any event, as discussed above, Hanan et al (1996), Solomon (Fisher 1998), and the attached Solomon declaration, clearly show that 10D5 inhibited aggregation of Aβ.

In paragraph 28 of the Official action, the examiner cites Pan et al for teaching that anti-Aβ antibodies, i.e., 3D6, decreases plaques in PDAPP mice by decreasing the concentration of Aβ in the central nervous system, not by disaggregation. Thus, the examiner contends that Pan et al teaches that Aβ plaques are not disassembled or prevented *per se*, but their formation is inhibited or in another sense slowed.

First of all, the experimentation reported in Pan was conducted on normal ICR mice, and not the PDAPP Alzheimer's disease mouse model. Thus, these mice do not spontaneously form amyloid plaques in the absence of antibody. The fact that the antibodies were shown to decrease the influx of Aβ into the brain does not necessarily mean that plaque is decreased.

Pan et al provides evidence that 3D6 can reduce the blood-to-brain influx of Aβ. However, this is merely one possible mechanism of action of 3D6. Pan et al does not exclude other mechanisms of action of 3D6. Indeed other mechanisms of action were not even tested in Pan et al. In this regard, Pan et al teaches, at page 614:

> Thus, we have shown that peripherally administered antibodies can decrease the availability of blood-borne Aβ to the brain. This does not rule out other routes of action, such as direct penetration of the antibody into the CNS or an influence on the solubility and CSF dynamics of Aβ ... In addition the N-terminal epitope (1-28) of Aβ is essential for aggregation (21) and the 3-6 sequential epitope is particularly important (8, 9). mAb3D6 is directed to the 1-5 sequence and likely prevented the aggregation of Aβ. (Emphases added)

Hence, contrary to the examiner's contention, Pan et al presents no experimental evidence on the issue of plaque disassembly or prevention, although the above quote does not rule out the possibility of such routes of action, i.e., disassembly or prevention of plaque.

In this regard, the examiner is requested to note that DeMattos et al (2001) states that 10D5 and 3D6, which are

effective at suppressing Aβ deposition *in vivo* in PDAPP mice are also able to decrease the concentration of Aβ in the central nervous system, i.e., act as Aβ sinks. Thus, this reference concludes that disaggregation is one mechanism of inhibition of Aβ aggregation that contributes to the effects of peripherally administered anti-amyloid antibodies, and that they can not exclude the possibility that antibodies, such as 266, enter the brain and sequester a soluble, toxic Aβ species.

In any event, the claims have been amended (new claims 150-167) to recite "inhibition" of aggregation, thereby rendering moot this aspect of the examiner's rejection.

In paragraph 29 of the Official action, the examiner cites Akiyama et al for teaching that 6F/3D does not readily bind plaques in cerebral cortex sample from an Alzheimer's patient.

However, this result is entirely consistent with the data and teachings in the Solomon application and Solomon (PNAS 1996), which shows that 6F/3D does not prevent aggregation, and teaches that 6F/3D does not bind to a disaggregation epitope. Applicant respectfully draws the examiner's attention to the fact that 6F/3D tests negative in the Solomon experiments and is, therefore, not covered by the claims. Note, Akiyama et al teaches, at page 328, right-hand column, that extracellular deposits retain immunoreactivity of N-terminal residues.

In paragraph 30 of the Official action, the examiner cites Perutz et al as showing the structure of amyloid fibers, and as providing the basis for the examiner's belief that anti-Aβ antibodies may inhibit or slow aggregation, but do not disassemble aggregates.

As discussed above, the data in Solomon (PNAS 1997) clearly demonstrate disaggregation of Aβ aggregates.

In any event, in view of the amendments to the claims (new claims 150-167) that recite "inhibition" of aggregation, applicant respectfully submits that the examiner's rejection has been rendered moot.

For all of these reasons, reconsideration and withdrawal of this rejection is respectfully urged.

The present specification has now been amended to correct an obvious error in the first paragraph of column 7. The patent stated that in a preferred embodiment the expression vector includes the sequence for a human monoclonal antibody "that is an anti-β-amyloid monoclonal antibody with heparan-like characteristics." The reference to "heparan-like characteristics" is nonsensical. The only reference to heparan in the specification is as an aggregating agent (column 11, lines 27-29, and column 16, lines 9-12). The antibodies inhibit aggregation of β-amyloid in the presence or absence of heparan sulfate. Thus, the antibodies do not have "heparan-like characteristics." To correct this obvious error, the words "with heparan-like characteristics" have now been deleted from this paragraph.

It should further be noted that column 16, lines 5-9, of the patent state:

> Binding of mAb AMY-33 to βA4 prevents self-aggregation of the β-amyloid, probably by recognizing the sequence 25-28 located in the proposed aggregation fragment comprising the amino acids between 25-28 (Yankher et al., 1990)(FIG.8).

It is not presently believed that the epitope of AMY-33 is the sequence 25-28 of β-amyloid. However, the above quote only indicates that it "probably" recognizes this sequence. Therefore, there is no necessity to correct it. The present statement, however, clarifies the record in this regard.

Copies of all publications cited herein that are not already of record or attached to the Solomon declaration are attached hereto.

It is submitted that all of the claims now present in the case clearly define the references of record. Reconsideration and allowance are therefore earnestly solicited.

Respectfully submitted,

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CLAIM SUPPORT CHART

CLAIM	SUPPORT
Claim 150. A pharmaceutical formulation, comprising:	C. 9, L. 23-25: It is preferable to present it as a **pharmaceutical formulation**. The **formulations** of the present inventions comprise….
(A) an **antibody** or **antigen binding fragment** thereof, wherein:	C. 5, L. 30-33: The **antibodies**, or peptide mimicking the binding site, must bind to an epitope on the target molecule which is a region responsible for folding or aggregation. C. 9, L. 24-26: The formulations of the present invention comprise … **the monoclonal antibody** …. C. 9, L. 45-48: [T]he use of engineered **monoclonal antibodies and their fragments** … can be used in the present invention. C. 12, L. 1-8: Alternatively, commercially available **antibodies** can be used….A **polyclonal**, affinity purified rabbit IgG obtained against the synthetic Alzheimer β-amyloid. C. 16, L. 26-31: Recent advances in antibody engineering technology, as well as in the development of suitable delivery systems…make it possible to develop **functional** small **antibody fragments** to serve as therapeutic chaperones for the treatment of Alzheimer's disease….
(i) said antibody and said fragment **recognize an epitope within residues 1-28 of beta-amyloid**, and	C. 5, L. 30-33: The antibodies, or peptide mimicking the binding site, must bind to an **epitope** on the target molecule which is

CLAIM	SUPPORT
	a region responsible for folding or aggregation. **C. 6, L. 23-27:** In a further preferred embodiment the monoclonal antibody is an anti-β-amyloid and is designated AMY-33 which **recognizes amino acids 1-28 of β-amyloid.** **C. 15, L. 35-38:** mAb AMY-33…**raised against peptide[s]** …**1-28**…of the **β-amyloid.** **C. 15, L. 43-46:** The antibody AMY-33, which is supposed to recognize **an epitope spanned between sequence 1-28,** inhibits the β-amyloid aggregation
(ii) said antibody and said fragment **inhibit aggregation of beta-amyloid;** and	**C. 6, L. 21-23:** In the preferred embodiment the human monoclonal antibody that binds to an aggregating protein and **which prevents aggregation** is utilized. **C. 9, L. 61-62: The antibodies effect on the inhibition of aggregation ….** **C. 15, L. 43-46:** The antibody AMY-33, which is supposed to recognize an epitope spanned between sequence 1-28, **inhibits the β-amyloid aggregation….**
(B) a **pharmaceutically acceptable carrier.**	**C. 9, L. 24-27:** The formulations of the present invention comprise at least one active ingredient: the monoclonal antibody or expression vector together with one or more **pharmaceutically acceptable carriers….**
Claim 151. The pharmaceutical formulation of claim 150,	See claim 150
wherein said antibody is a **monoclonal antibody.**	**C. 5, L. 51-53:** In the preferred embodiment of the

CLAIM	SUPPORT
	method, the target molecule is β-amyloid and the **monoclonal antibody** is an anti-β-amyloid **monoclonal**. **C. 6, L. 1-6:** Once an appropriate **monoclonal antibody** with chaperone-like activity is found or engineered..., the present invention provides for its use therapeutically to prevent or reduce protein aggregation in vivo. **C. 9, L. 22-28:** It is preferable to present it as a pharmaceutical formulation. The formulations of the present invention comprise at least one active ingredient: **the monoclonal antibody** or expression vector together with one or more pharmaceutical acceptable carriers and optionally other therapeutic ingredients.
Claim 152. The pharmaceutical formulation of claim 151,	See claim 151
wherein said antibody is a **human monoclonal antibody**.	**C. 6, L. 21-23:** In the preferred embodiment the **human monoclonal antibody** that binds to an aggregating protein and which prevents aggregation is utilized. **C. 7, L. 7-12:** In a preferred embodiment the expression vector includes the sequence for a **human monoclonal antibody** that is an anti-β-amyloid monoclonal antibody with heparin-like characteristics.
Claim 153. The pharmaceutical formulation	See claim 151

CLAIM	SUPPORT
of claim 151,	
wherein said antibody is a **genetically-engineered monoclonal antibody.**	**C. 9, L. 45-48:** the use of **engineered monoclonal antibodies** and their fragments, as well as peptides which mimic the binding site for the antigen on the antibody can be used in the present invention. **C. 10, L. 1-5:** The present invention uses **genetically engineered antibodies** obtained from such selected antibodies as protecting agents of in vivo aggregation of their antigen….

Claim 154. The pharmaceutical formulation of claim 153,	See claim 153
wherein said antibody is a **single-chain antibody.**	**C. 6. L. 27-29:** Work by Duenas et al. (1994) and Marasco et al. (1993) have shown that **single chain monoclonal antibodies** are efficient for intracellular expression in eukaryotes. **C. 7, L. 9-11:** In a further preferred embodiment, the expression vector includes the sequence for the **single chain monoclonal antibody** of the above anti-β-amyloid mAb. **C. 16, L. 34-37:** Application of the above findings for in vivo aggregation, can confer to **single chain antibodies** or other engineered antibody fragments, a protective role in the renaturation of recombinant proteins

Claim 155. The pharmaceutical formulation of any one of claims 150-154,	See claims 150-154

CLAIM	SUPPORT
wherein said beta-amyloid is **human beta-amyloid.**	**C. 8, L. 19-21:** The expression vector containing the sequence for the anti-aggregation molecule may be administered to mammals, including **humans…** **C. 11, L. 20-23:** Amyloid peptides, Aβ 1-40 (Cat. No. A-5813) and Aβ 1-28 (Cat. No. A-1084) corresponding to amino acids 1-40 and 1-28 of Aβ respectively, were produced from Sigma Chemical Co., St. Louis, MO., USA. **C. 12, L. 1-3:** Alternatively, commercially available antibodies can be used. **α-Human β-amyloid** 6F/3D was obtained…. **C. 16, L. 27-33:** Recent advances in antibody engineering technology, as well as in the development of suitable delivery systems…make it possible to develop functional small antibody fragments to serve as therapeutic chaperones for the treatment of **Alzheimer's disease as well as other human amyloidosis diseases….**

Claim 156. A pharmaceutical formulation, comprising:	**C. 9, L. 23-25:** It is preferable to present it as a **pharmaceutical formulation.** The **formulations** of the present inventions comprise….
(A) an **antibody** or **antigen binding fragment** thereof, wherein:	**C. 5, L. 30-33:** The **antibodies,** or peptide mimicking the binding site, must bind to an epitope on the target molecule which is a region responsible for folding or aggregation. **C. 9, L. 24-26:** The formulations of the present invention comprise … **the**

CLAIM	SUPPORT
	monoclonal antibody …. C. 9, L. 45-48: [T]he use of engineered monoclonal antibodies and their fragments … can be used in the present invention. C. 12, L. 1-8: Alternatively, commercially available antibodies can be used….A polyclonal, affinity purified rabbit IgG obtained against the synthetic Alzheimer β-amyloid. C. 16, L. 26-31: Recent advances in antibody engineering technology, as well as in the development of suitable delivery systems…make it possible to develop functional small antibody fragments to serve as therapeutic chaperones for the treatment of Alzheimer's disease….
(i) said antibody is obtainable using residues 1-28 of beta-amyloid as an immunogen, and	C. 6, L. 23-27: In a further preferred embodiment the monoclonal antibody is an anti-β-amyloid and is designated AMY-33 which recognizes amino acids 1-28 of β-amyloid. C. 15, L. 35-38: mAb AMY-33…raised against peptide[s] …1-28…of the β-amyloid. C. 15, L. 43-46: The antibody AMY-33, which is supposed to recognize an epitope spanned between sequence 1-28, inhibits the β-amyloid aggregation….
(ii) said antibody and said fragment inhibit aggregation of beta-amyloid; and	C. 6, L. 21-23: In the preferred embodiment the human monoclonal antibody that binds to an aggregating protein and which prevents aggregation is utilized. C. 9, L. 61-62: The

CLAIM	SUPPORT
	antibodies effect on the inhibition of aggregation …. **C. 15, L. 43-46:** The antibody AMY-33, which is supposed to recognize an epitope spanned between sequence 1-28, **inhibits the β-amyloid aggregation….**
(B) a **pharmaceutically acceptable carrier.**	**C. 9, L. 24-27:** The formulations of the present invention comprise at least one active ingredient: the monoclonal antibody or expression vector together with one or more **pharmaceutically acceptable carriers….**

Claim 157. The pharmaceutical formulation of claim 156,	See claim 156
wherein said antibody is a **monoclonal antibody.**	**C. 5, L. 51-53:** In the preferred embodiment of the method, the target molecule is β-amyloid and the **monoclonal antibody** is an anti-β-amyloid **monoclonal.** **C. 6, L. 1-6:** Once an appropriate **monoclonal antibody** with chaperone-like activity is found or engineered…, the present invention provides for its use therapeutically to prevent or reduce protein aggregation in vivo. **C. 9, L. 22-28:** It is preferable to present it as a pharmaceutical formulation. The formulations of the present invention comprise at least one active ingredient: **the monoclonal antibody** or expression vector together with one or more pharmaceutical acceptable carriers and optionally other

CLAIM	SUPPORT
	therapeutic ingredients.
Claim 158. The pharmaceutical formulation of claim 157,	See claim 157
wherein said antibody is a **human monoclonal antibody.**	**C. 6, L. 21-23:** In the preferred embodiment the **human monoclonal antibody** that binds to an aggregating protein and which prevents aggregation is utilized. **C. 7, L. 7-12:** In a preferred embodiment the expression vector includes the sequence for a **human monoclonal antibody** that is an anti-β-amyloid monoclonal antibody with heparin-like characteristics.
Claim 159. The pharmaceutical formulation of claim 157,	See claim 157
wherein said antibody is a **genetically-engineered monoclonal antibody.**	**C. 9, L. 45-48:** the use of **engineered monoclonal antibodies** and their fragments, as well as peptides which mimic the binding site for the antigen on the antibody can be used in the present invention. **C. 10, L. 1-5:** The present invention uses **genetically engineered antibodies** obtained from such selected antibodies as protecting agents of in vivo aggregation of their antigen….
Claim 160. The pharmaceutical formulation of claim 159,	See claim 159
wherein said antibody is a **single-chain antibody.**	**C. 6. L. 27-29:** Work by Duenas et al. (1994) and Marasco et al. (1993) have shown that **single chain monoclonal antibodies** are

CLAIM	SUPPORT
	efficient for intracellular expression in eukaryotes. **C. 7, L. 9-11:** In a further preferred embodiment, the expression vector includes the sequence for the **single chain monoclonal antibody of** the above anti-β-amyloid mAb. **C. 16, L. 34-37:** Application of the above findings for in vivo aggregation, can confer to **single chain antibodies** or other engineered antibody fragments, a protective role in the renaturation of recombinant proteins
Claim 161. The pharmaceutical formulation of any one of claims 156-160,	See claims 156-160
wherein said beta-amyloid is **human beta-amyloid.**	**C. 8, L. 19-21:** The expression vector containing the sequence for the anti-aggregation molecule may be administered to mammals, including **humans…** **C. 11, L. 20-23:** Amyloid peptides, Aβ 1-40 (Cat. No. A-5813) and Aβ 1-28 (Cat. No. A-1084) corresponding to amino acids 1-40 and 1-28 of Aβ respectively, were produced from Sigma Chemical Co., St. Louis, MO., USA. **C. 12, L. 1-3:** Alternatively, commercially available antibodies can be used. **α-Human β-amyloid** 6F/3D was obtained…. **C. 16, L. 27-33:** Recent advances in antibody engineering technology, as well as in the development of suitable delivery systems…make it possible to develop functional small

CLAIM	SUPPORT
	antibody fragments to serve as therapeutic chaperones for the treatment of **Alzheimer's disease as well as other human amyloidosis diseases**….
Claim 162. A pharmaceutical formulation, comprising:	**C. 9, L. 23-25**: It is preferable to present it as a **pharmaceutical formulation**. The **formulations** of the present inventions comprise….
(A) an **antibody** or **antigen binding fragment** thereof, wherein:	**C. 5, L. 30-33**: The **antibodies**, or peptide mimicking the binding site, must bind to an epitope on the target molecule which is a region responsible for folding or aggregation. **C. 9, L. 24-26**: The formulations of the present invention comprise … **the monoclonal antibody** …. **C. 9, L. 45-48**: [T]he use of engineered **monoclonal antibodies and their fragments** … can be used in the present invention. **C. 12, L. 1-8**: Alternatively, commercially available **antibodies** can be used….A **polyclonal**, affinity purified rabbit IgG obtained against the synthetic Alzheimer β-amyloid. **C. 16, L. 26-31**: Recent advances in antibody engineering technology, as well as in the development of suitable delivery systems…make it possible to develop **functional** small **antibody fragments** to serve as therapeutic chaperones for the treatment of Alzheimer's disease….
(i) said antibody and said fragment **recognize an**	**C. 5, L. 30-33**: The antibodies, or peptide

CLAIM	SUPPORT
epitope within residues 1-28 of beta-amyloid, and	mimicking the binding site, must bind to an **epitope** on the target molecule **which is a region responsible for folding or aggregation.** **C. 6, L. 23-27:** In a further preferred embodiment the monoclonal antibody is an anti-β-amyloid and is designated AMY-33 which **recognizes amino acids 1-28 of β-amyloid.** **C. 15, L. 35-38:** mAb AMY-33...**raised against peptide[s] ...1-28...**of the **β-amyloid.** **C. 15, L. 43-46:** The antibody AMY-33, which is supposed to recognize **an epitope spanned between sequence 1-28,** inhibits the β-amyloid aggregation
(ii) said antibody and said fragment **maintain the solubility of soluble beta-amyloid;** and	**C. 1., L. 35-37:** In vitro **aggregation limits** the protein stability, **solubility** and yields in production of recombinant proteins. **C. 3, L. 54-56:** which **prevents aggregation** and allows biological activity of the target molecule. **C. 6, L. 12-15:** ...binds to an aggregating protein which is the cause of a disease and which **prevents aggregation** and yet allows the protein to be bioactive. **Col. 10, L. 1-5:** The present invention uses genetically engineered antibodies obtained from such selected antibodies of in vivo aggregation of their antigen, leading to production of a **soluble and stabilized protein.** **Col. 10, L. 16-19:** The identification of such

CLAIM	SUPPORT
	classes of sequences that play a role in the folding-unfolding and/or **solubilization**-aggregation provides the basis of the present invention for prevention of aggregation. **C. 13, L. 30-32, 38-40, Fig. 7a and 7b:** The residual **soluble β-amyloid** was incubated for another one hour at 37° C with mAbs AMY-33 and/or 6F3D at equal molar ratio antibody/antigen….**The amount of mAb bound will be proportional to the amount of soluble amyloid which remained after exposure to aggregating conditions.**
(B) a **pharmaceutically acceptable carrier.**	**C. 9, L. 24-27:** The formulations of the present invention comprise at least one active ingredient: the monoclonal antibody or expression vector together with one or more **pharmaceutically acceptable carriers….**

Claim 163. The pharmaceutical formulation of claim 162,	See claim 162
wherein said antibody is a **monoclonal antibody.**	**C. 5, L. 51-53:** In the preferred embodiment of the method, the target molecule is β-amyloid and the **monoclonal antibody** is an anti-β-amyloid **monoclonal. C. 6, L. 1-6:** Once an appropriate **monoclonal antibody** with chaperone-like activity is found or engineered…, the present invention provides for its use therapeutically to prevent or reduce protein aggregation in vivo.

CLAIM	SUPPORT
	C. 9, L. 22-28: It is preferable to present it as a pharmaceutical formulation. The formulations of the present invention comprise at least one active ingredient: **the monoclonal antibody** or expression vector together with one or more pharmaceutical acceptable carriers and optionally other therapeutic ingredients.

Claim 164. The pharmaceutical formulation of claim 163,	See claim 163
wherein said antibody is a **human monoclonal antibody.**	C. 6, L. 21-23: In the preferred embodiment the **human monoclonal antibody** that binds to an aggregating protein and which prevents aggregation is utilized. C. 7, L. 7-12: In a preferred embodiment the expression vector includes the sequence for a **human monoclonal antibody** that is an anti-β-amyloid monoclonal antibody with heparin-like characteristics.

Claim 165. The pharmaceutical formulation of claim 163,	See claim 163
wherein said antibody is a **genetically-engineered monoclonal antibody.**	C. 9, L. 45-48: the use of **engineered monoclonal antibodies** and their fragments, as well as peptides which mimic the binding site for the antigen on the antibody can be used in the present invention. C. 10, L. 1-5: The present invention uses **genetically engineered antibodies** obtained from such selected

CLAIM	SUPPORT
	antibodies as protecting agents of in vivo aggregation of their antigen….
Claim 166. The pharmaceutical formulation of claim 165,	See claim 165
wherein said antibody is a **single-chain antibody.**	**C. 6. L. 27-29:** Work by Duenas et al. (1994) and Marasco et al. (1993) have shown that **single chain monoclonal antibodies** are efficient for intracellular expression in eukaryotes. **C. 7, L. 9-11:** In a further preferred embodiment, the expression vector includes the sequence for the **single chain monoclonal antibody** of the above anti-β-amyloid mAb. **C. 16, L. 34-37:** Application of the above findings for in vivo aggregation, can confer to **single chain antibodies** or other engineered antibody fragments, a protective role in the renaturation of recombinant proteins
Claim 167. The pharmaceutical formulation of any one of claims 162-166,	See claims 162-166
wherein said beta-amyloid is **human beta-amyloid.**	**C. 8, L. 19-21:** The expression vector containing the sequence for the anti-aggregation molecule may be administered to mammals, including **humans…** **C. 11, L. 20-23:** Amyloid peptides, Aβ 1-40 (Cat. No. A-5813) and Aβ 1-28 (Cat. No. A-1084) corresponding to amino acids 1-40 and 1-28 of Aβ respectively, were produced from Sigma Chemical